Available online at www.scholarsresearchlibrary.com

Scholars Research Library

Scholars Research Library

Annals of Biological Research, 2011, 2 (6) :36-41
(http://scholarsresearchlibrary.com/archive.html)

Annals of Biological Research
Scholars Research Library
ISSN 0976-1233
CODEN (USA): ABRNBW

Eosinophils and Mast Cells in Leishmaniasis

[1]Eilyad Issabeagloo*, [2]Parviz Kermanizadeh, [3]Farhad Ahmadpoor, [4]Mohammad Taghizadieh

[1]Department of Pharmacology, Medical Sciences Faculty, Tabriz branch, Islamic Azad University, Tabriz, Iran
[2]Department of Internal Medicine, Medical Sciences Faculty, Tabriz branch, Islamic Azad University, Tabriz, iran
[3]Department of Basic Sciences, Medical Sciences Faculty, Tabriz branch, Islamic Azad University, Tabriz, Iran
[4]Department of Pathology, Medical Sciences Faculty, Tabriz branch, Islamic Azad University, Tabriz, Iran

---

ABSTRACT

Lesions in the spleen and livers of Leishmania infantum -infected BALB/C were studied for 36 weeks and the frequency of mast cells and eosinophils in the lesions were calculated. The tissues were stained with a newly developed method designed to detect mast cells and eosinophils simultaneously. Lesions appeared from week 13 post-infection and both the number and size of the lesions increased gradually and continued as the infection matured. Eosinophils comprised (95%) of the cell types in the lesions and probably play an important role in Leishmania infantum control. In comparison to eosinophils less number of the mast cells was observed in the lesions all the times during the infection.

Key words: *leishmania infantum*, Eosinophils, Mast cells,

INTRODUCTION

Leishmania a protozoan parasite that lives primarily within macrophages, causes the disease leishmaniasis. *Leishmania donovani* causes visceral leishmaniasis (VL), disseminates to spleen, liver and bone marrow (BM), but L. major causes cutaneous leishmaniasis (CL), remains in the cutaneous lesion and the draining lymph node (1)

The resistance or susceptibility of the host depends on the selection of Th1 or Th2 lymphocyte and activity that seems to operate in most infections and leishmaniasis models as well. Susceptibility of inbred mouse strains to *Leishmania* infection is attributed to the predominance of the Th2 cytokine pattern response which is not strong among the various mouse strains considered resistant (2).

CD4-Th2 cells (the main sources of IL-4 and IL-13) are responsible for production of mast cells, basophiles, and eosinophils (3-4). Which are recruited into peripheral sites to take part in innate immunity as the effector cells against local stimuli (5)

Upon stimulation mast cells and eosinophils produce various bioactive mediators, such as histamine, arachidonic acid metabolites, proteases, chemokines, and cytokines rapidly (5, 6). Also CD4-Th2 cells are the main sources of IL-4 and IL-13, non-lymphoid producers of these cytokines also may play an important role in immunity, pathology (7, 8) of parasitic infections (9, 10)

MATERIALS AND METHODS

A total of 110 female BALB/C mice 8-10 weeks old (maintained in our laboratory) were infected with *L infantum*, by injecting of 1000 promastigotes in the peritoneum.

Four infected & two control mice were processed each 10 days during 36 weeks investigation for histology study. Liver and spleen tissue was fixed in MT* fixative for 24 h , tissues were processed and embedded in paraffin. 5µm thick sections were cut and stained with a new method (11).

At least ten sections were measured in each tissue and were subjected to cell counting for every sample of liver and spleen tissues. The number of mast cells and eosinophils were counted in each section by x 400 magnification (per mm^2). The total number of lesions in a minimum of 10 graticules of each infected section was calculated.

RESULTS

From week 3 post infection eosinophils were observed in the liver and spleen samples, the skin samples showed a little number of eosinophils. By week 10 post infection clusters of eosinophils could be seen, predominantly in peri-vascular areas of the liver and spleen tissue. At week 13, lesions started to form and rapidly became prominent in tissue sections. The number of lesions in the liver increased from 18 per 100 mm^2 at week 13 to 70 per 100 mm^2 at week 36 and the number of lesions in the spleen increased from 10 per 100 mm^2 at week 25 to 30 per 100 mm^2 at week 36, end of the experiment (Fig.1).

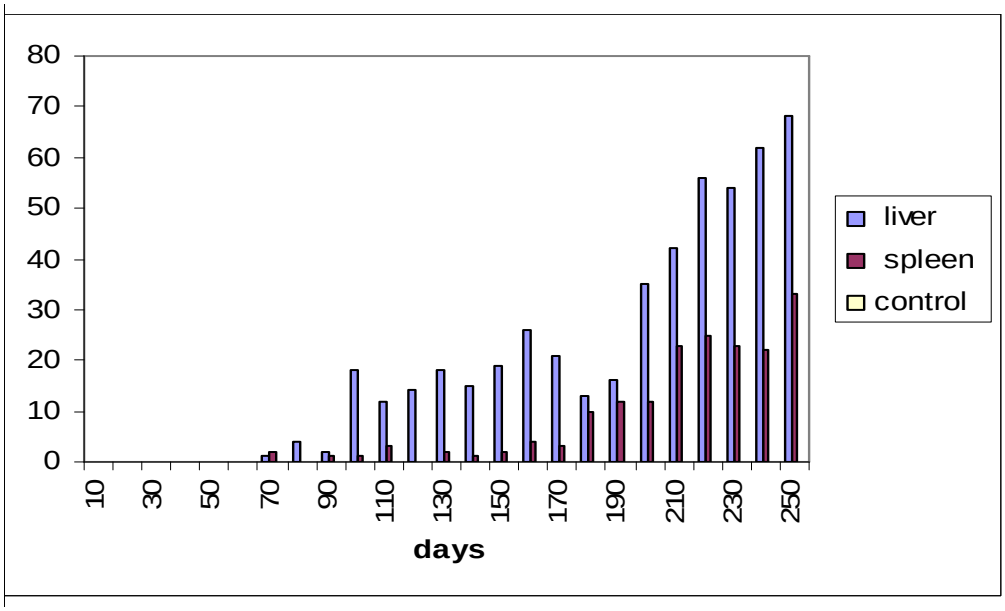

Figure 1: lesions in liver and spleen

The number of eosinophils in the liver was seen to be high ranging from 101 per mm^2 at week 4 post-infection to 4600 per mm^2 by week 36 and the number of eosinophils in the spleen was seen ranging from 16 per mm^2 at week 3 post-infection to 1560 per mm^2 by week 36 (Fig.2, 3).

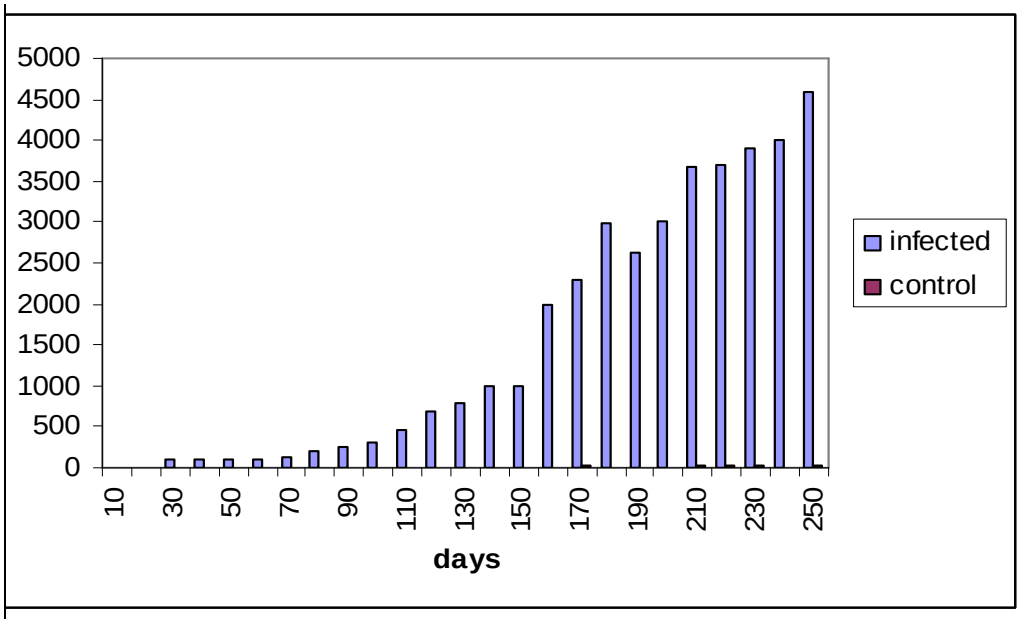

Figure 2: amount of Eosinophils in liver

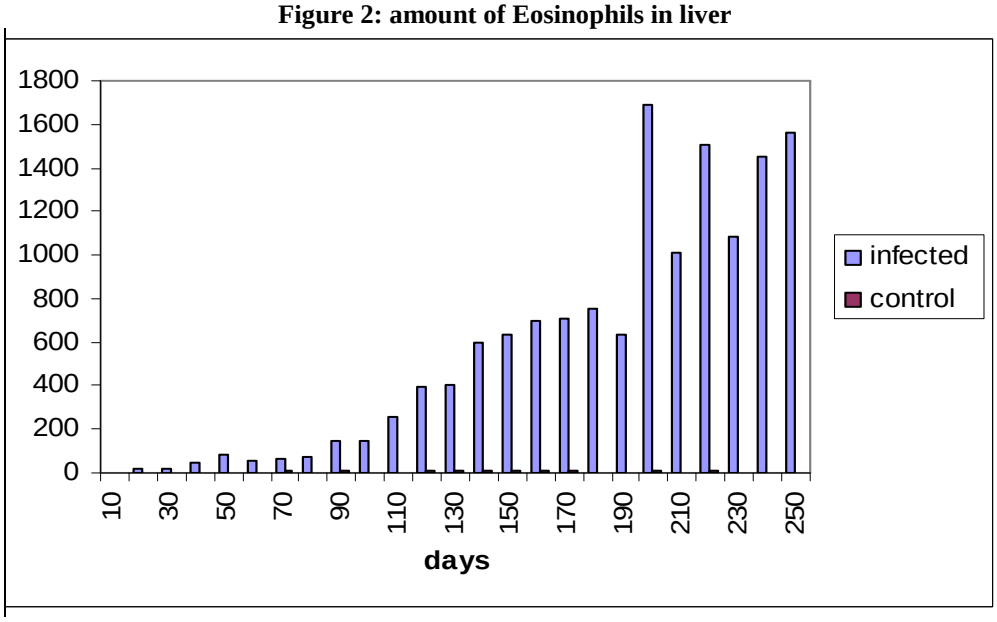

Figure 3: amount of Eosinophils in spleen

Mast cells were observed in liver tissue by week 5 post-infection. The number of the mast cells increased from 11 per mm^2 at week 5 post infection reaching a maximum of 230 per mm^2 at week 36 and the number of the mast cells in the spleen increased from 23-34 per mm^2 at week 30 post infection and reducing to 11 per mm^2 later end of the experiment (Fig.4,5). Compared with the number of the eosinophils, mast cells comprised only a small per cent of the total cell number in the infected animals.

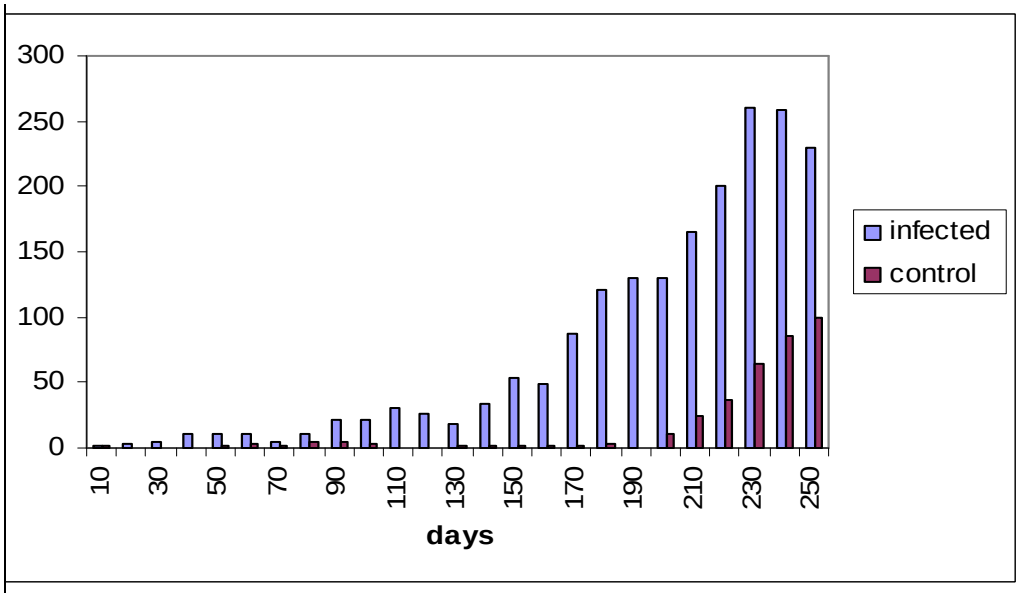

Figure 4: amount of Mast cells in liver

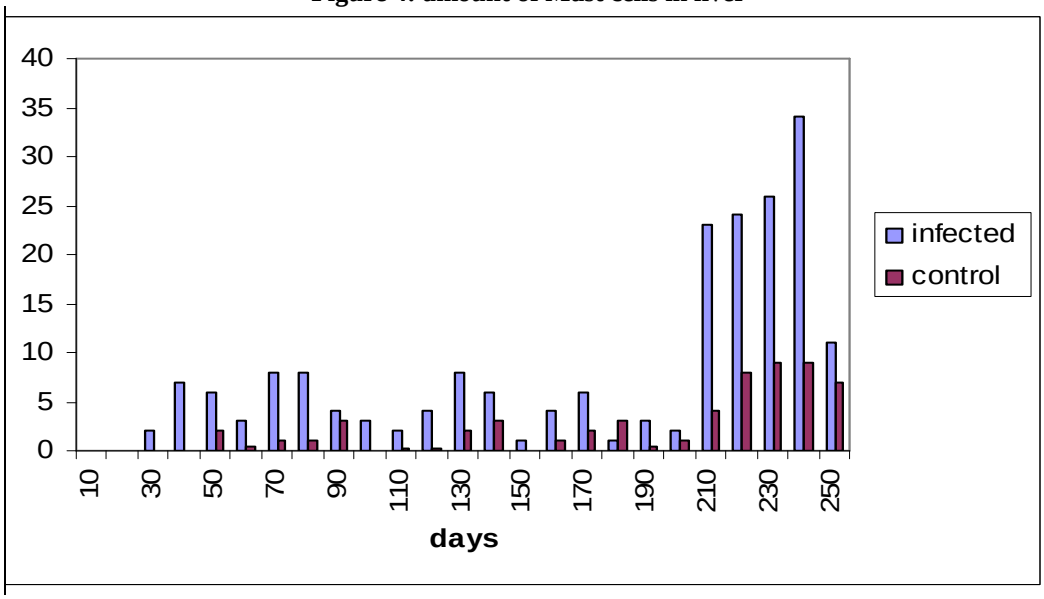

Figure 5: amount of Mast cells in spleen

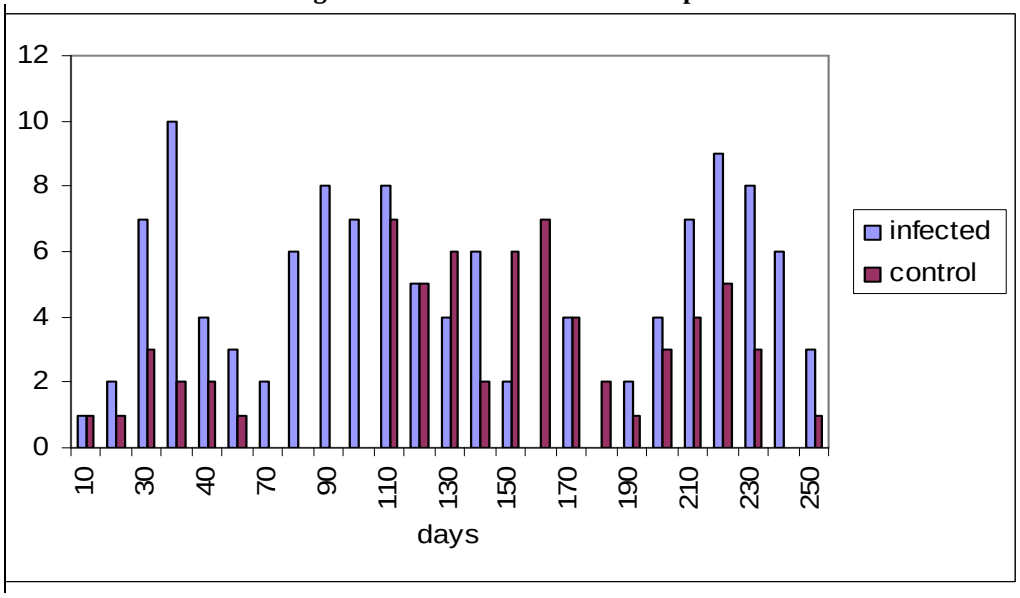

Figure 6: amount of Eosinophils in skin

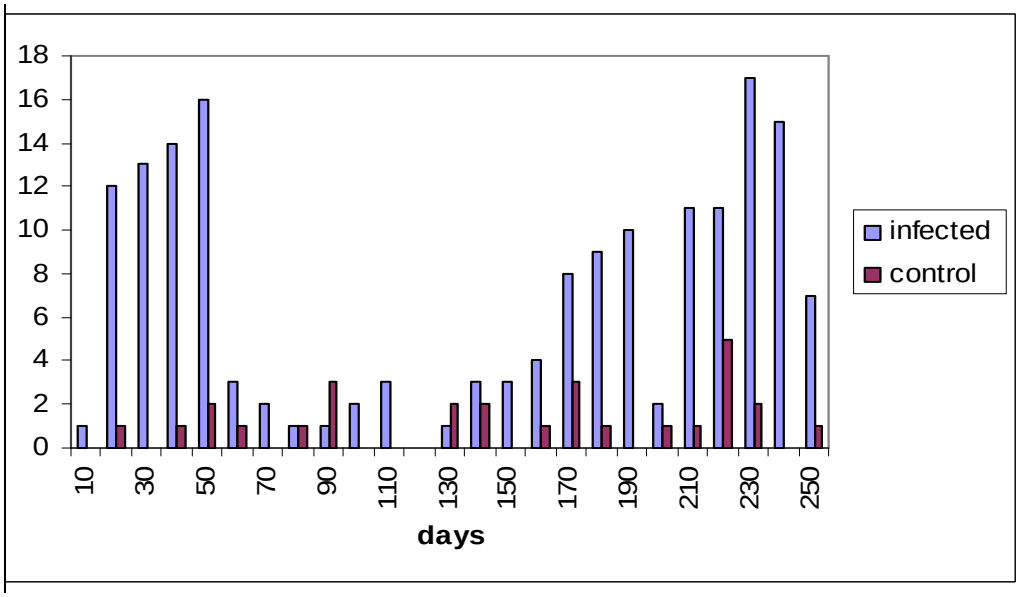

Figure 7: amount of Mast cells in skin

The number of mast cells in the skin increased from 12-16 per mm^2 at week 3-7 and 11-15 per mm^2 at week 30-34 (Fig.6, 7). There was no big difference in eosinophils number in the skin of the infected and control animals

DISCUSSION

Mast cells and eosinophils are bone marrow derived cells are collectively regarded as key effectors of type2 immunity and immunopathology (12). Despite their different development, homing properties, diversity of effector functions, and the phenotypic heterogeneity of mast cells, they share the potential for IL-4 and IL-13 production upon stimulation (13, 14). Mast cells and eosinophils, moreover, are preferentially located in peripheral inflammatory sites and are positioned to mediate effector functions and orchestrate type2 immunity (15, 16). According to the results that obtained in the current study, lesion numbers increased from 13 weeks post-infection and reached a peak by 36 weeks post-infection. This peak at 36 weeks post-infection may be the time of parasite dissemination. Eosinophils numbers were observed to increase after week 4 and their number remained high throughout the experiment. Unlike eosinophils, mast cell numbers were seen to increase from week 5 post-infection, reaching a maximum 230 per mm2 by week 36 post-infection. 4.7% of the cells present in the lesions were mast cells and 95.2 % were eosinophils. One interpretation is that whilst the eosinophils may be an effector cell actively participating in destruction the mast cell is more important in maintaining control of tissue repair and regeneration and may therefore have a helping role in fibroblast activation, regulating their activity in collagen production (17, 18). The mast cell dynamics fit with the developing of lesions, were frequently observed towards the periphery of the lesions and always followed the increase in eosinophils numbers. intracellular micro-organisms such as Leishmania, have adopted many different mechanisms for their replication inside the host (19) and the host resistance is depend on the development of specific cell-mediated immunity (20,21). Among other cells eosinophils which have crucial role in cell cytotoxicity have been reported to participate to the control of parasitic infections (22, 23). Also the precise mechanisms of Leishmania destruction remain to be established. But following mechanisms of eosinophils may be important in parasites destruction Firstly: Eosinophils may lyses infected phagocytes, secondly: reactive oxygen intermediates released by eosinophils might be responsible for the parasite destruction (24), thirdly: eosinophils could function as immunoregulatory cells by

releasing soluble mediators such as TNFα (25, 26) that regulate the entry and intracellular multiplication of parasite in host cells.

REFERENCES

[1] Luc Nicolas., Sacha Sidjanski., Jean-Herve ' Colle and Genevie' Ve Milon. *Infection and immunity.* **2000**, 68: 6561–6566
[2] F. D. Finkelman, T. Shea-Donohue, J. Goldhill, C. A. Sullivan, S. C. Morris, K. B. Madden, W. C. Gause, and J. F. Urban, *Jr. Annu. Rev. Immunol*. **1997**, 15:505.
[3] F. D. Finkelman, T. Shea-Donohue, J. Goldhill, C. A. Sullivan, S. C. Morris, and K. B. Madden, W. C. Gause, and J. F. Urban, *Jr. Annu. Rev. Immunol*. **1997**, 15:505.
[4] T. Kawakami, and S. J. Galli. *Nat. Rev. Immunol*. **2002**. 2:773.
[5] S. J. Galli, M. Maurer, and C. S. Lantz. *Curr. Opin. Immunol*. **1999**, 11:53.
[6] G. M. Walsh. *Curr. Opin. Hematol*. **2001**, 8:28.
[7] M. Wills-Karp. *Annu. Rev. Immunol*. **1999**, 17:255.
[8] M. A. Brown, and J. Hural. *Crit. Rev. Immunol*. **1997**, 17:1.
[9] B. de Andres, E. Rakasz, M. Hagen, M. L. McCormik, A. L. Mueller, D. Elliot, A. Metwali, M. Sandor, B. E. Britigan, J. V. Weinstock, et al. *Blood*. **1997**, 89:3826.
[10] A.E. Butterworth, *Adv. Parasitol*., **1984**, 23,143-235
[11] P. Kermanizadeh, P. Hagan & D.W.T. Crompton, *Parasitology Today*, **1995**, 11, 194-196.
[12] M. Wills-Karp, Annu. Rev. Immunol, **1999**, 17:255.
[13] P. Bardding, J.A. Roberts, K.M. Britten, S. montefort, R. Dgukanovic, R. Mueller, C.H. Heusser, P.H. Howarth and S. Holgate, *Am .J.Respir. Cell Mol.Biol*, **1994**, 10: 471-480.
[14] J.S. Jaffe, D.G. Raible, T.J. Post, Y. Wang, M.C. Glaum, J.H. Butterfield, and E.S. Schulman, *Am. J. Respir. Cell Mol. Biol*, **1996**, 15: 473-481.
[15] B. S. Bochner, and R. P. Schleimer, *Immunol. Rev*, **2001**, 179:5.
[16] K. Shinkai, M. Mohrs, and R. M. Locksley, *Nature*, **2002**, 420:825.
[17] A. Schittek, A.A. Demetriou, J. Padawar, *Agents Actions*, **1984**, 15: 172-176.
[18] D.W. Fitzpatrick, H. Ficsher, *Surgery*, **1982**, 97: 430-434.
[19] C. Bogdan, M. Rollinghoff, *Int J Parasitol*, **1998**, 28:121–134.
[20] F.P. Heinzel, M.D. Sadick, B.J. Holaday, *J Exp Med*,**1989**, 169:59 -72.
[21] H.W. Murray, H. Masur, J.S. Keithl, *J Immunol*, **1982**, 129:344–349.
[22] K. Hirokazu, David A. Loegering, Cheryl R. Adolphson, Gerald J. Gleich *Int Arch Allergy Immunol*, **1999**, 118:426-428
[23] M.Capron, J. Rousseaux, C. Mazingue, H. Bazin & A. Capron, *The Journal of Immunology*, **1978**, 121, 2518-2525.
[24] R. E. Aldridge, T. Chan, C. J. van Dalen, R. Senthilmohan, M. Winn, *Archives of Biochemistry and Biophysics*, **2006**, 445: 235-24.
[25] S. Finotto, I. Ohno, *J Immunol*, **1994**, 153(5):2278-89.
[26] J. J. Costa, K. Matossian, M. B. Resnick, W. J. Bell, D. T. W. Wong, J. R. Gordon, A. M. Dvorak, P. F. Weller & S. J. Galli, Journal of Clinical Investigation, **1993**, 91, 2673-2684.